was sclerosis of the posterior root-zones of the cord. In the case under consideration there was no trace of disease of any portion of the posterior columns, proving that, for the production of ataxy, degeneration of the cord was not an essential factor. Reference was made to the cases published by M. Déjerine, in which there were all the symptoms of tabes, and after death only parenchymatous neuritis of the peripheral nerve-endings found, the cord-roots being intact. From this it was maintained that interruption of the different paths, at the periphery, was capable of causing ataxy in as characteristic a manner as when these tracts were diseased in the cord. The case under notice showed that lesion of a third locality, namely, of the posterior spinal roots, might produce the same effects. It was asserted that this was a pathological confirmation of the experimental researches of Van Deen and Claude Bernard, who, by dividing the posterior roots, induced inco-ordination of movements without motor paralysis. It supported the view that those elements which conveyed the impulses regulating co-ordination, were situated in the afferent paths of the nervous system. Whatever theory be advanced to explain the physiology of locomotor ataxy, this case served to show that the point at which, in the causation of the phenomena, the nervous path was interrupted, must not of necessity, as was generally asserted, be primarily situated in the posterior root-zones of the spinal cord. Reflection on this fact suggested that the anatomical substratum of that protean disorder which was at present recognized under the term tabes dorsalis, had not yet been fathomed. The assemblage of symptoms probably consisted of a combination of different pathological conditions, many of which were represented by phenomena common to all, and each of which in time might be distinguished. It was possible that a case such as the present might prove as a link in the chain of discovery.—*Brit. Med. Jour.*, No. 1,262.

Clonic Spasms of Upper Limbs.—Dr. C. E. Beevor read a paper before the Medical Society, London, on clonic spasm of the upper limbs, with anæsthesia of the left side, in a girl aged 16, who had scarlet-fever ten years before she came under observation, and, six months after the fever, began to have twitching of the head and face to the left, and movements in both arms. She came under treatment by Dr. Barlow for choreiform movements affecting all the limbs seven years ago; movements had persisted ever since. A year ago, the left arm alone was affected with pronation, extension of the elbow, and adduction of the shoulder, but voluntary movements were possible ; she also had occasional rapid twisting of the head, and both eyes were turned to the left. In October, 1884, the right arm was affected in a similar way, and the left arm became anæsthetic and analgesic. When the patient was shown at the meeting, the movements of the left arm were much quieter, but the limb was almost completely paralyzed, and

anæsthetic and analgesic. The anæsthesia also affected the whole left side of the trunk, head, and neck,—except the face, which was only somewhat anæsthetic—down to the umbilical level. Below this level there was a less degree of anæsthesia. In the area of complete anæsthesia, touch, pain, heat and cold, and sensibility to a strong faradic wire-brush were all abolished. In the conjunctiva, nostril, mouth, tongue, and fauces, on the left side, sensibility was deficient. In the left arm there was complete loss of muscular sense to weights and passive movement. Sight was not affected, nor the field of vision. The other senses were not affected, and there was no optic neuritis. In the arms, the muscles affected corresponded, according to Ferrier's and Yeo's experiments, to the sixth and seventh cervical nerves. The case was probably an instance of a functional or hysterical nature. The movements were irregularly rhythmical, about sixty to eighty per minute, ceasing during sleep; and, though simulating chorea, were much more regular, and affected only certain muscles; the movements of one arm were synchronous, but the two arms did not move as a rule at the same time.—*Brit. Med. Jour.*, No. 1,265.

Nervous Lesions by Lead-Poisoning.—Arthur Robinson, M.B.,C.M., (from Path. Lab. Owen's Col., England, *Brain*, xxviii., p. 485,) reports a case of lead paralysis with autopsy. (Dr. Leech's case, Manchester Royal Infirmary). A male, æt., 30; a painter for fourteen years, had his first attack of drop-wrist in the summer of 1877. It continued through the winter, recovering the following spring. Every winter after, the attack returned; continuing until spring. He never had colic or cramps, but was troubled by frequent micturition. When examined, in December, 1882, the muscles of both forearms were very much atrophied, the extensors of the wrists and fingers were completely paralyzed, while the flexors retained a fair amount of power. The muscles of the thenar and hypothenar eminences were also atrophied, and the patient could not oppose his thumbs. He could pronate, but could only partially supinate both forearms. The peroneus brevis and longus muscles on the left side were atrophied and paralyzed, and those of the right side were affected with paresis. All the atrophied muscles showed the reaction of degeneration when tested with electricity. The patient was treated with iodide of potassium and the interrupted and continuous currents were applied to the affected muscles. He gradually improved; the atrophied muscles increased in size, and to a certain extent regained their power. Discharged much improved, in 1883, he returned in June, 1884, his muscular status much the same, but with œdema of the extremities, pleuritic effusion, albuminous urine, and granular casts; later ascites developed, and death occurred August, 1884. The autopsy revealed small kidneys with adherent capsule; and, on microscopical examination advanced cirrhosis. The spinal cord and parts of the musculo-spiral nerves